

What is claimed is:

1. A method for activating interferon regulatory factor 3 (IRF3) in a cell comprising contacting the cell with a molecule that stimulates a Toll-like receptors (TLR), thereby activating the IRF3 in the cell, wherein the cell expresses the TLR.
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2. The method of claim 1, wherein the TLR is TLR3.
3. The method of claim 1, wherein the TLR is TLR4.
- 10 4. The method of claim 1, wherein the molecule that binds the TLR is a TLR ligand selected from a group consisting of bacterial antigen, LPS, lipid A, taxol, viral antigen, RSV F protein, double stranded RNA, imidazoquinoline compounds, and poly I:C.
- 15 5. A method for inhibiting a microbial infection comprising contacting the cell with a molecule that stimulates induction of IRF3 in the cell, thereby inhibiting the microbial infection.
- 20 6. The method of claim 5, wherein the inhibition of microbial infection is effected by inducing expression of primary response genes.
7. The method of claim 5, wherein the molecule that stimulates induction of IRF3 in the cell is a molecule that stimulates a TLR.
- 25 8. The method of claim 7, wherein the TLR is TLR3.
9. The method of claim 7, wherein the TLR is TLR4.
- 30 10. The method of claim 7, wherein the molecule that stimulates the TLR is a TLR ligand selected from a group consisting of a bacterial antigen, LPS, lipid A, taxol, a viral

antigen, RSV F protein, double stranded RNA, imidazoquinoline compounds, and poly I:C.

11. The method of claim 6, wherein the primary response protein is any of IFIT1, ISG15,
5 RANTES, IP10, or IFN β .

12. The method of claim 6, wherein the microbial infection is a viral, fungal or bacterial infection.

10 13. A method for inhibiting a microbial infection by inducing activity of IFN β in a cell comprising contacting the cell with a molecule that stimulates induction of IRF3.

14. The method of claim 13, wherein expression of IFN β activates STAT1, thereby inducing activity of a secondary response protein in a cell.

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15. The method of claim 14, wherein the secondary response protein is any one of Mx1, IFI1, IFI204, or IRF7.

16. The method of claim 13, wherein the microbial infection is a viral, fungal or bacterial
20 infection.

17. The method of claim 13, wherein the molecule that stimulates activity of IRF3 is a TLR ligand selected from a group consisting of bacterial antigen, LPS, lipid A, taxol, viral antigen, RSV F protein, double stranded RNA, imidazoquinoline compounds, and poly I:C.
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18. A method for inhibiting a microbial infection by stimulating induction of any one of MX1, IFI1, IFI204, IRF7, IFT3, IRG1, IRF9, IFI-TM31, PKR, EBI3, or IFN α 5 in a cell comprising contacting the cell with a molecule that stimulates induction of IFN β ,
30 thereby stimulating induction of MX1, IFI1, IFI204, IRF7, IFT3, IRG1, IRF9, IFI1, IFI-TM31, PKR, EBI3, or IFN α 5, so as to inhibit the microbial infection in the cell.

19. The method of claim 18, wherein the molecule that stimulates activity of IFN β is a TLR ligand selected from a group consisting of bacterial antigen, LPS, lipid A, taxol, viral antigen, RSV F protein, double stranded RNA, imidazoquinoline compounds, and poly I:C.
20. A method for inhibiting viral replication in a cell by stimulating the TLR3/TLR4 and IRF3 pathways in the cell comprising contacting the cell with a molecule that stimulates the TLR3/TLR4 and IRF3 pathways, thereby inhibiting the viral replication in the cell.
21. The method of claim 20, wherein the molecule that stimulates the TLR3/TLR4 and IRF3 pathways is a TLR ligand selected from a group consisting of bacterial antigen, LPS, lipid A, taxol, viral antigen, RSV F protein, double stranded RNA, imidazoquinoline compounds, and poly I:C.
22. A method for inducing anti-inflammatory response in a cell by suppressing the TLR3/TLR4 and IRF3 pathways in the cell comprising contacting the cell with a molecule that suppresses the TLR3/TLR4 and IRF3 pathways, thereby inducing an anti-inflammatory response in the cell.
23. The method of claim 22, wherein the molecule that suppresses the TLR3/TLR4 and IRF3 pathways is a TLR ligand selected from a group consisting of a soluble TLR, an anti-TLR antibody, an anti-interferon antibody, an anti-LPS antibody, and molecules that block endotoxin shock.
24. A method for identifying small molecules that inhibit a microbial infection by activating any of the genes selected from a group consisting of MX1, IFI1, IFI204, and IRF7 in a cell, the method comprising contacting a cell with a molecule of interest that binds a TLR and activates the genes thereby inhibiting a microbial infection..